FÜR: WST VON: HJA DATUM: 07.12.92
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Datenbankanbieter: DIALOG

File Nr.	Name	Zeitraum:	Bemerkungen:
File 154:	MEDLINE_1985-1992/DEC (9212W4)		
File 55:	BIOSIS PREVIEWS_85-92/DEC BA9412:BARRM4312		
File 156:	TOXLINE _ 1965-1992/NOV		
File 72:	EMBASE (EXCERPTA MEDICA)_85-92/ISS49		

Suchtabelle **(Suche in allen Files gleichzeitig):**

Set	Items	Description
S1	652276	MOUSE OR MICE
S2	344	HEAD(1W)ONLY
S3	715	NOSE(1W)ONLY
S4	143	S1 AND (S2 OR S3)
S5	72	RD S4 (unique items)

5/AU,TI,SO,DE,AB/1 (Item 1 from file: 154)
AU- Bellossi A
TI- Effect of pulsed magnetic fields on healthy mice, a study through cellular electrophoresis of thymic cells.
SO- <JN> Acta Med Hung
SO- <VO> 48 (1-2) p95-102
SO- <PY> 1991
DE- <MINOR> Cell Separation --Methods --MT; Electrophoresis --Methods --MT; Mice; Oxidopamine; Reference Values; Thymus Gland --Cytology --CY; Time Factors; Wakefulness
DE- <MAJOR> *Magnetics; *Peripheral Nerves --Physiology --PH; *Thymus Gland --Radiation Effects --RE
AB- An exposure of adult mice to a pulsed magnetic field (PMF) increases thymus weight. In this work, thymic lymphocytes were studied by cellular electrophoresis because the proliferation and maturation of these cells is linked to an increase in their electrophoretic mobility (EM). Fifteen-week-old female Swiss mice were exposed for 30 min to a 6 mT PMF, 12 or 460 Hz in frequency, according to different modalities. The EM of the thymic cells, suspended in saline were measured from 0 to 96 h after the end of the exposure. For some of the mice the whole body, for others the head only or the body without the head was exposed; the animals were awaken or narcotized, prepared or not with 6-hydroxydopamine. The modifications of the EM are in favour of an action of the PMF on the thymus through the central nervous system.

5/AU,TI,SO,DE,AB/2 (Item 2 from file: 154)
AU- Gairola CG; Wagner GJ
TI- Cadmium accumulation in the lung, liver and kidney of mice and rats chronically exposed to cigarette smoke.
SO- <JN> J Appl Toxicol
SO- <VO> 11 (5) p355-8

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SO- <PY> Oct 1991
DE- <MINOR> Administration, Inhalation; Body Weight --Drug Effects --DE; Mice; Mice, Inbred C57BL; Rats; Rats, Inbred Strains; Time Factors
DE- <MAJOR> *Cadmium --Pharmacokinetics --PK; *Kidney --Metabolism --ME; *Liver --Metabolism --ME; *Lung --Metabolism --ME; *Smoke --Adverse Effects --AE
AB- Cigarette smoking constitutes a major source of cadmium exposure via inhalation in man. To determine how smoke exposure affects the organ distribution and accumulation of cadmium, male C57B1 mice and Sprague-Dawley rats were exposed daily for 52-60 consecutive weeks to mainstream smoke from the University of Kentucky reference cigarettes (2R1) in a nose-only exposure system. Exposed mice and rats averaged blood carboxyhemoglobin values of 17.7 and 7.2%, and a daily total particulate matter (TPM) dose of 7.2 and 3.2 mg kg-1 body wt. per exposure, respectively. These results suggested effective inhalation of smoke by the animals. The tissues were acid digested and analyzed for cadmium by atomic absorption spectrometry. Cadmium levels 5-6- and 2-3-fold greater than control levels were detected in the lungs and kidneys, respectively, of exposed animals of both species. In contrast, the liver did not show increased cadmium levels in exposed mice or rats. The data suggest that low-dose chronic inhalation exposure to cigarette smoke leads to highest cadmium accumulation in the lung, followed by the kidney, with minimal effects on cadmium levels in the liver.

5/AU,TI,SO,DE,AB/3 (Item 3 from file: 154)
AU- McAnulty RJ; Moores SR; Talbot RJ; Bishop JE; Mays PK; Laurent GJ
TI- Long-term changes in mouse lung following inhalation of a fibrosis-inducing dose of 239PuO2: changes in collagen synthesis and degradation rates.
SO- <JN> Int J Radiat Biol
SO- <VO> 59 (1) p229-38
SO- <PY> Jan 1991
DE- <MINOR> Administration, Inhalation; Collagen --Biosynthesis --BI Collagen --Metabolism --ME; Mice; Mice, Inbred CBA; Plutonium --Administration and Dosage --AD; Time Factors
DE- <MAJOR> *Collagen --Radiation Effects --RE; *Lung --Radiation Effects --RE; *Plutonium; *Pulmonary Fibrosis --Etiology --ET
AB- Mice were exposed by nose-only inhalation to 239PuO2, which resulted in an IAD of 1110 +/- 29 Bq. At various times after exposure, rates of collagen metabolism were measured using validated in vivo methods based on the administration of radiolabelled proline, together with a large flooding dose of unlabelled proline and measurement of its incorporation into lung collagen as hydroxyproline. Dramatic increases in both synthesis and degradation rates of collagen were observed. At 54 days after exposure the fractional synthesis rates in experimental mice were almost five times those in controls (control: 3.2 +/- 0.6%/day, 239PuO2-exposed: 14.5 +/- 0.4%/day) and by 300 days synthesis rates, although declining, were still more than double the control values. A similar pattern of change was observed for collagen degradation. The combination of changes in synthesis and degradation rates led to a 60% increase in lung collagen content by 300 days (control: 3.05 +/- 0.24 mg/lung, 239PuO2-exposed: 4.88 +/- 0.42 mg/lung). The data suggest that extensive remodelling of the lung connective tissue matrix occurs during development of fibrosis and that, over long periods of time, small imbalances between synthesis and degradation may result in quite large increases in protein content.

5/AU,TI,SO,DE,AB/4 (Item 4 from file: 154)

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AU- Barrow DM; Blehm KD
TI- A predictive model for vapor concentration in a nose-only inhalation chamber.
SO- <JN> Am Ind Hyg Assoc J
SO- <VO> 51 (9) p494-9
SO- <PY> Sep 1990
DE- <MINOR> Methyl N-Butyl Ketone --Analysis --AN; Mice; Nose; Occupational Medicine --Instrumentation --IS; Spectrophotometry; Volatilization
DE- <MAJOR> *Environmental Exposure; *Gases; *Models, Theoretical; *Nebulizers and Vaporizers
AB- A unique nose-only inhalation chamber was designed and constructed to deliver uniform concentrations of gas, vapor, and aerosol contaminants to mice. This research investigated the fluid dynamics of a vaporous contaminant in the vertical flow chamber. The vapor was introduced by allowing the liquid phase of the contaminant to evaporate freely into the chamber interior. A contaminant mass transfer model was developed to predict concentrations generated by the system. The mathematical model of the system used clean airflow, liquid surface area, thickness of the stagnant air layer covering the liquid, system pressure, contaminant diffusion coefficient, and contaminant vapor pressure to compute the vapor concentration delivered to exposure ports. The equation was verified by placing various containers of methyl isobutyl ketone in the chamber and determining with a photospectrometer the resulting equilibrium concentrations. Vapor pressure, diffusion coefficient, and system pressure were held constant while airflow, surface area, and stagnant air layer thickness were varied systematically within the chamber. The resulting empirical data points were compared to the curves predicted by the theoretical model. Empirical concentrations fell within 0 to 48% of the theoretical values, showing that the equation can be used to choose values for airflow, surface area, and stagnant air layer thickness that will result in chamber concentrations in close proximity to the target concentration. If an exact concentration is essential, parameters may be individually adjusted to converge on the target concentration.

5/AU,TI,SO,DE,AB/5 (Item 5 from file: 154)
AU- Sabourin PJ; Sun JD; MacGregor JT; Wehr CM; Birnbaum LS; Lucier G; Henderson RF
TI- Effect of repeated benzene inhalation exposures on benzene metabolism, binding to hemoglobin, and induction of micronuclei.
SO- <JN> Toxicol Appl Pharmacol
SO- <VO> 103 (3) p452-62
SO- <PY> May 1990
DE- <MINOR> Administration, Inhalation; Benzene --Administration and Dosage --AD Benzene --Toxicity --TO; Carbon Radioisotopes --Diagnostic Use --DU; Drug Administration Schedule; Environmental Exposure; Erythrocytes --Drug Effects --DE Erythrocytes --Physiology --PH; Genes --Drug Effects --DE; Glucuronates --Urine --UR; Hydroquinones --Urine --UR; Rats; Time Factors
DE- <MAJOR> *Benzene --Metabolism --ME; *Hemoglobins --Metabolism --ME; *Micronuclei --Drug Effects --DE
AB- Metabolism of benzene is thought to be necessary to produce the toxic effects, including carcinogenicity, associated with benzene exposure. To extrapolate from the results of rodent studies to potential health risks in man, one must know how benzene metabolism is affected by species, dose, dose rate, and repeated versus single exposures. The purpose of our studies was to determine the effect of repeated inhalation exposures on the metabolism of [14C]benzene by rodents. Benzene metabolism was assessed by characterizing and quantitating

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urinary metabolites, and by quantitating 14C bound to hemoglobin and micronuclei induction. F344/N rats and B6C3F1 mice were exposed, nose-only, to 600 ppm benzene or to air (control) for 6 hr/day, 5 days/week for 3 weeks. On the last day, both benzene-pretreated and control animals were exposed to 600 ppm, 14C-labeled benzene for 6 hr. Individual benzene metabolites in urine collected for 24 hr after the exposure were analyzed. There was a significant decrease in the respiratory rate of mice (but not rats) pretreated with benzene which resulted in lower levels of urinary [14C]benzene metabolites. The analyses indicated that the only effects of benzene pretreatment on the metabolite profile in rat or mouse urine were a slight shift from glucuronidation to sulfation in mice and a shift from sulfation to glucuronidation in rats. Benzene pretreatment also had no effect, in either species, on formation of [14C]benzene-derived hemoglobin adducts. Mice and rats had similar levels of hemoglobin adduct binding, despite the higher metabolism of benzene by mice. This indicates that hemoglobin adduct formation occurs with higher efficiency in rats. After 1 week of exposure to 600 ppm benzene, the frequency of micronucleated, polychromatic erythrocytes (PCEs) in mice was significantly increased. Exposure to the same level of benzene for an additional 2 weeks did not further increase the frequency of micronuclei in PCEs. These results indicate that repeated exposures to benzene, such as might be encountered by humans as a result of occupational or environmental exposures, are not likely to change or increase benzene metabolism.

5/AU,TI,SO,DE,AB/6 (Item 6 from file: 154)
AU- Wathes CM; Zaidan WA; Pearson GR; Hinton M; Todd N
TI- Aerosol infection of calves and mice with Salmonella typhimurium.
SO- <JN> Vet Rec
SO- <VO> 123 (23) p590-4
SO- <PY> Dec 3 1988
DE- <MINOR> Aerosols; Cattle; Humidity; Mice; Mice, Inbred BALB C; Salmonella typhimurium --Isolation and Purification --IP; Salmonella Infections, Animal --Microbiology --MI
DE- <MAJOR> *Air Microbiology; *Cattle Diseases --Transmission --TM; *Salmonella typhimurium --Growth and Development --GD; *Salmonella Infections, Animal --Transmission --TM
AB- The likelihood of airborne spread and infection by Salmonella typhimurium was studied in calves and mice. S typhimurium survived in air sufficiently long to present a significant hazard of airborne spread. In a dry climate (32 per cent relative humidity) its predicted viability five minutes after aerosolization was 4 per cent relative to its initial value. This increased to 24.8 per cent in a humid atmosphere (72 per cent relative humidity). Inhalation of S typhimurium by mice caused disease and death at times dependent upon the dose. Even the lowest dose of about 150 colony forming units (cfu) produced disease. Inoculation of approximately 10(4) to 10(6) cfu S typhimurium by either 'mouth and nose only' or 'whole body' aerosol exposure caused infection in calves. The consequences for the control of salmonellosis in intensive calf husbandry are discussed in the light of this demonstration that airborne transmission can be a primary mode of infection of S typhimurium.

5/AU,TI,SO,DE,AB/7 (Item 7 from file: 154)
AU- Bond JA; Martin OS; Birnbaum LS; Dahl AR; Melnick RL; Henderson RF
TI- Metabolism of 1,3-butadiene by lung and liver microsomes of rats and mice repeatedly exposed by inhalation to 1,3-butadiene.
SO- <JN> Toxicol Lett
SO- <VO> 44 (1-2) p143-51

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SO- <PY> Nov 1988
DE- <MINOR> Administration, Inhalation; Chromatography, Gas; Mice; Microsomes --Metabolism --ME; Rats; Rats, Inbred Strains; Species Specificity
DE- <MAJOR> *Butadienes --Metabolism --ME; *Lung --Metabolism --ME; *Microsomes, Liver --Metabolism --ME
AB- 1,3-Butadiene, a colorless gas widely used as an intermediate in the production of synthetic rubber, is carcinogenic in rats and mice. Species differences exist in the sensitivity to inhaled 1,3-butadiene and the target tissue specificity for tumor formation. We examined whether repeated inhalation exposure of rats and mice to 1,3-butadiene would affect the rate of metabolism of 1,3-butadiene by lung and liver microsomes in these species. Male Sprague-Dawley rats and B6C3F1 mice were exposed nose-only to air (control) or 7600 +/- 170 ppm 1,3-butadiene (13,600 +/- 300 micrograms/l) and 740 +/- 10 ppm 1,3-butadiene (1300 +/- 20 micrograms/l), respectively, for 6 h/day for 5 days. After the last exposure, nasal tissue (rats only), lungs and livers were removed from the animals and microsomes were prepared. Microsomes from the different tissues were incubated with 6 mumol 1,3-butadiene and 10 mumol NADPH for 30 min and the rate of disappearance of 1,3-butadiene from the reaction flasks was quantitated. There was a statistically significant (P less than 0.05) depression in the rate of 1,3-butadiene metabolism (50%) in microsomes from lungs of both rats and mice that were exposed repeatedly to 1,3-butadiene compared to control animals. There was no effect of repeated 1,3-butadiene exposure on liver or nasal tissue (rats only) metabolism of 1,3-butadiene in rats or mice. The data from these studies indicate that it is unlikely that species differences in sensitivity or tissue susceptibility are due to an inductive or inhibitory effect of 1,3-butadiene on its own metabolism in the tissues examined.

5/AU,TI,SO,DE,AB/8 (Item 8 from file: 154)
AU- Cunningham MJ; Choy WN; Arce GT; Rickard LB; Vlachos DA; Kinney LA; Sarrif AM

TI- In vivo sister chromatid exchange and micronucleus induction studies with 1,3-butadiene in B6C3F1 mice and Sprague-Dawley rats.
SO- <JN> Mutagenesis
SO- <VO> 1 (6) p449-52
SO- <PY> Nov 1986
DE- <MINOR> Administration, Inhalation; Bone Marrow --Drug Effects --DE Bone Marrow --Ultrastructure --UL; Cell Nucleus --Drug Effects --DE; Erythrocytes --Drug Effects --DE Erythrocytes --Ultrastructure --UL; Mice; Mutagenicity Tests --Methods --MT; Rats; Rats, Inbred Strains; Sister Chromatid Exchange --Drug Effects --DE; Species Specificity
DE- <MAJOR> *Butadienes --Toxicity --TO; *Mutagens
AB- Male B6C3F1 mice and Sprague-Dawley rats were exposed for 2 days, 6 h/day to 1,3-butadiene (BD) by inhalation (nose only) and their bone marrow cells were evaluated for the induction of micronuclei (MN) and sister chromatid exchanges (SCEs). A significant dose-dependent increase in MN induction was observed in mice. At 100 p.p.m., the frequency of micronucleated polychromatic erythrocytes was 6-fold above control with a maximal induction of 38-fold at 10,000 p.p.m. A significant increase in SCEs was also observed in mouse bone marrow cells starting at 100 p.p.m. with a 4-fold increase over the control evident at 10,000 p.p.m. The highest tested no observed effect level for both endpoints was 50 p.p.m. In contrast, rat bone marrow cells did not exhibit significant increases in micronucleated polychromatic erythrocytes or SCEs. These results indicate that BD is genotoxic in the bone marrow of the mouse but not the rat. This paralleled the

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chronic bioassays which showed mice to be more susceptible than rats to BD carcinogenicity.

5/AU,TI,SO,DE,AB/9 (Item 9 from file: 154)
AU- Stephenson EH; Moeller RB; York CG; Young HW
TI- Nose-only versus whole-body aerosol exposure for induction of upper respiratory infections of laboratory mice.
SO- <JN> Am Ind Hyg Assoc J
SO- <VO> 49 (3) p128-35
SO- <PY> Mar 1988
DE- <MINOR> Disease Models, Animal; Encephalomyelitis, Venezuelan Equine --Microbiology --MI Encephalomyelitis, Venezuelan Equine --Pathology --PA Encephalomyelitis, Venezuelan Equine --Transmission --TM; Klebsiella pneumoniae; Klebsiella Infections --Microbiology --MI Klebsiella Infections --Pathology --PA Klebsiella Infections --Transmission --TM; Lethal Dose 50; Mice; Nose; Respiratory Tract Infections --Microbiology --MI Respiratory Tract Infections --Pathology --PA
DE- <MAJOR> *Aerosols; *Respiratory Tract Infections --Transmission --TM
AB- The effectiveness of two aerosol delivery systems, nose-only and whole-body, were compared using Swiss-Webster mice and two pathogens, Klebsiella pneumoniae and Venezuelan equine encephalitis (VEE) virus. With K. pneumoniae the median lethal dose (LD50) and the mean time to death correlated with the inhaled dose. An LD50 value of 335 colony forming units (cfu) for nose-only exposure was significantly less than the LD50 value of 3741 cfu obtained for whole-body exposure. The LD50 values obtained with VEE virus for nose-only exposure [8 plaque forming units (pfu)] and whole-body exposure (11 pfu) were similar to each other. Following a 10-min nose-only exposure, concentrations of K. pneumoniae approximating 10(4)/g were present after 24 hr in the upper respiratory tract (URT) and lungs. The numbers of bacteria reached a peak at 72 hr, when resolution of the infection began. Detectable levels of bacteria in the blood and tissues were delayed in mice given whole-body exposure, plus there was a decreased concentration of bacteria per gram of tissue. Major pathological lesions induced by K. pneumoniae were mild suppurative rhinitis and minimal suppurative bronchopneumonia. Viremia was greatest at 96 hr following aerosol exposure to VEE. Virus concentrations in the URT, lungs, cerebrum, spleen and mesenteric lymph nodes reached maximum titers earlier for mice exposed by nose-only than for mice exposed to whole-body aerosols.(ABSTRACT TRUNCATED AT 250 WORDS)

5/AU,TI,SO,DE,AB/10 (Item 10 from file: 154)
AU- Kaplan HL; Anzueto A; Switzer WG; Hinderer RK
TI- Effects of hydrogen chloride on respiratory response and pulmonary function of the baboon.
SO- <JN> J Toxicol Environ Health
SO- <VO> 23 (4) p473-93
SO- <PY> 1988
DE- <MINOR> Atmosphere Exposure Chambers; Carbon Dioxide --Blood --BL; Oxygen --Blood --BL; Papio; Tidal Volume
DE- <MAJOR> *Hydrochloric Acid --Toxicity --TO; *Lung --Drug Effects --DE
AB- The effects of hydrogen chloride (HCl) inhalation on respiratory response during exposure and on pulmonary function during the 3 mo following exposure were studied in the baboon. Each of 4 groups of three anesthetized animals was exposed in a head-only mode for 15 min to air or one of three HCl concentrations (500, 5000, or 10,000 ppm). The acute respiratory response consisted of a concentration-related increase in frequency and minute volume, with a marked decrease in blood PaO2 at the two highest concentrations. The exposures did not

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cause significant alterations in any of the pulmonary function parameters measured at 3 d and 3 mo postexposure. Thus, nonhuman primates were able to survive short exposures to high concentrations of HCl without any significant effects on pulmonary function during the 3 mo after exposure. Furthermore, comparison of the response of primates and rodents suggests that the human is much less sensitive to the effects of HCl than the mouse.

5/AU,TI,SO,DE,AB/11 (Item 11 from file: 154)
AU- Bond JA; Dahl AR; Henderson RF; Birnbaum LS
TI- Species differences in the distribution of inhaled butadiene in tissues.
SO- <JN> Am Ind Hyg Assoc J
SO- <VO> 48 (10) p867-72
SO- <PY> Oct 1987
DE- <MINOR> Administration, Intranasal; Butadienes --Toxicity --TO; Mice; M
ice, Inbred Strains; Rats; Rats, Inbred Strains; Tissue Distribution
DE- <MAJOR> *Butadienes --Pharmacokinetics --PK
AB- 1,3-Butadiene is produced commercially for use in the manufacture of elastomers, polymers and other chemicals. Recent inhalation carcinogenicity studies of butadiene indicate that B6C3F1 mice are more sensitive to the tumorigenic effects of inhaled butadiene than are Sprague Dawley rats. The purpose of this investigation was to determine if there were differences in distribution in tissues of inhaled butadiene between rats and mice. Male Sprague Dawley rats and B6C3F1 mice were exposed nose-only for 3.4 hr to (mean +/- SE) 1220 +/- 71 micrograms 14C-butadiene/L air and 121 +/- 2 micrograms 14C-butadiene/L air, respectively. Radioactivity was distributed widely in tissues immediately following exposure of both rats and mice to 14C-butadiene. In both species, respiratory tract tissue (lung, trachea, nasal turbinates), gastrointestinal tract (small and large intestine), liver, kidneys, urinary bladder and pancreas contained high concentrations of radioactivity within 1 hr after the end of exposure. In all cases, tissues of mice contained 15 to 100 times the concentration of 14C-butadiene equivalents per mumole of butadiene inhaled than did rats. For both rats and mice, elimination of 14C from tissues and blood was rapid, with 77% to 99% of the initial tissue burden being eliminated with half-times of 2 to 10 hr. Within 1 hr after the end of exposure, all rat tissues retained a substantial amount of 14C that was associated with volatile material (20% to 40% of the total 14C in tissues) that was probably butadiene and/or metabolites. A similar observation was noted in mouse liver, the only tissue of mice examined.(ABSTRACT TRUNCATED AT 250 WORDS)

5/AU,TI,SO,DE,AB/12 (Item 12 from file: 154)
AU- Walk RA; Jenderny J; Rohrborn G; Hackenberg U
TI- Chromosomal abnormalities and sister-chromatid exchange in bone marrow cells of mice and Chinese hamsters after inhalation and intraperitoneal administration: I. Diepoxybutane.
SO- <JN> Mutat Res
SO- <VO> 182 (6) p333-42
SO- <PY> Dec 1987
DE- <MINOR> Administration, Inhalation; Cricetulus; Epoxy Compounds --Admin
istration and Dosage --AD; Hamsters; Injections, Intraperitoneal; Mice;
Species Specificity
DE- <MAJOR> *Chromosome Aberrations; *Epoxy Compounds --Pharmacology --PD;
*Ethers, Cyclic --Pharmacology --PD; *Sister Chromatid Exchange --Drug
Effects --DE
AB- Diepoxybutane (DEB), a direct-acting animal carcinogen, was found to increase the frequency of structural chromosomal abnormalities (CA) and

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sister-chromatid exchange (SCE) in bone marrow cells of mice and Chinese hamsters, when inhaled from an aerosol during a 2-h head-only exposure or administered as a single intraperitoneal injection. For the purpose of comparing the genotoxicity in the 2 species, both after inhalation and intraperitoneal administration, the systemic DEB dose obtained by inhalation was determined on the basis of blood concentrations and inhalation duration after the investigation of the blood kinetics. The bone marrow cells of male and female NMRI mice were found to be more sensitive than those of Chinese hamsters to the genotoxic activity of DEB.

5/AU,TI,SO,DE,AB/13 (Item 13 from file: 154)
AU- Sendelbach LE; Witschi HP
TI- Bronchoalveolar lavage in rats and mice following beryllium sulfate inhalation.
SO- <JN> Toxicol Appl Pharmacol
SO- <VO> 90 (2) p322-9
SO- <PY> Sep 15 1987
DE- <MINOR> Acid Phosphatase --Analysis --AN; Albumins --Analysis --AN; Alkaline Phosphatase --Analysis --AN; Irrigation; Lactate Dehydrogenase --Analysis --AN; Lung --Analysis --AN; Mice; Mice, Inbred BALB C; Rats; Rats, Inbred F344
DE- <MAJOR> *Beryllium --Toxicity --TO; *Lung --Drug Effects --DE
AB- The effects of lung injury in rats and mice exposed to an aerosol of beryllium sulfate for 1 hr through nose-only inhalation were evaluated by the method of bronchoalveolar lavage. The lavage fluid of rats exposed to an aerosol of either 3.3 or 7.0 micrograms Be/liter over a 21-day period following exposure indicated lactate dehydrogenase (LDH) and alkaline phosphatase (Alk Pase) activities to be the most sensitive indicators of lung damage. LDH activity peaked at 8 days postexposure while Alk Pase activity was maximum at Day 5. Both values were 30 times greater than comparable controls at these time points. Acid phosphatase activity and albumin levels also increased over the 21-day period, but not to the same extent. The lung lavage of mice exposed to 7.2 micrograms Be/liter showed LDH activity as the most sensitive indicator of lung damage with a maximum response three times greater than that of controls at Day 5.

5/AU,TI,SO,DE,AB/14 (Item 14 from file: 154)
AU- Yang VC; Ainsworth EJ
TI- A histological study on the cataractogenic effects of heavy charged particles.
SO- <JN> Proc Natl Sci Counc Repub China [B]
SO- <VO> 11 (1) p18-28
SO- <PY> Jan 1987
DE- <MINOR> Cataract --Pathology --PA; Dose-Response Relationship, Radiation; Lens, Crystalline --Pathology --PA; Mice; Mice, Inbred Strains; Radioisotopes; X-Rays
DE- <MAJOR> *Cataract --Etiology --ET; *Lens, Crystalline --Radiation Effects --RE; *Radiation Injuries, Experimental --Pathology --PA
AB- The morphology of the mouse lens was studied by light and electron microscopy between 4 and 30 months after graded single doses of photons or charged particle exposure. Four-month-old mice were given upper body or head only doses of 220 kVp x-rays or to 40Ar (570 MeV/amu), 40Ar(500 MeV/amu), 20Ne(470 MeV/amu) and 12C (400 MeV/amu) particles. The lenses examined in this pilot study were largely from mice dedicated to other designed experiments; nor all radiation conditions are represented at each sample time. At 4 months after 0.05, 0.3 or 0.9 Gy 20Ne radiation, the micronuclei appeared in the epithelial cells at the bow region. The anterior fibers showed swollen cytoplasm containing amphorous materials

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after Ø.3 or Ø.9 Gy 2ØNe exposure. The posterior fibers were relatively normal. The results at 15 months after irradiation indicated heavy charged particles were more effective than x-rays for inducing morphological changes; however there were no qualitative differences in damage produced by heavy charged particles and x-rays. Characteristic changes after irradiation included abnormal bow patterns, posterior and anterior migration of nucleated cells in the cortex, swelling of fibers and the development of swirls of epithelium at the anterior pole. The results also indicated that the degree of degenerative changes in lens fibers increases with increasing radiation dose and sampling time. The radiation-induced injury to lens fibers also showed a relationship to linear energy transfer (LET). The qualitative observations at 24 months indicated that low energy 4ØAr (6ØØ KeV/micron) is less effective than high energy 4ØAr particles (1ØØ KeV/micron)1 and radiation-induced injury in the lens fibers shows an LET-dependence when the ionizing density is less than 1ØØ KeV/micron. At 3Ø months after heavy charged particle irradiation, the changes in control lens were the swollen fibers, while in the 4ØAr-irradiated animals, aberrant fibers with abnormal nuclei, amorphous materials and debris were observed.

5/AU,TI,SO,DE,AB/15 (Item 15 from file: 154)
AU- Sendelbach LE; Witschi HP; Tryka AF
TI- Acute pulmonary toxicity of beryllium sulfate inhalation in rats and mice: cell kinetics and histopathology.
SO- <JN> Toxicol Appl Pharmacol
SO- <VO> 85 (2) p248-56
SO- <PY> Sep 15 1986
DE- <MINOR> Administration, Inhalation; Cell, Division --Drug Effects --DE; Kinetics; Lung --Pathology --PA; Macrophages --Pathology --PA; Mice; Mice, Inbred BALB C; Rats; Rats, Inbred F344; Species Specificity
DE- <MAJOR> *Beryllium --Toxicity --TO; *Lung --Drug Effects --DE
AB- The acute response in the lung of mice and rats exposed to an aerosol of BeSO4 was characterized by cell kinetics and histopathology. Animals were exposed for 1 hour in a nose-only chamber to a 13 microgram/liter chamber concentration of BeSO4 or to H2SO4 as control aerosol, and killed thereafter over a period of 21 days. Lung cell kinetics were evaluated as the labeling index (LI), defined as the percentage of cells labeled with tritiated thymidine. The labeled cells were also differentiated as to type. In rats, the LI showed a peak response on Day 8 following exposure, while the LI of mice showed a maximum response at Day 5. In rats, the proliferative response involved type II alveolar epithelial cells, interstitial and capillary endothelial cells. Histopathology showed type II alveolar cell hyperplasia and vacuolization of their cytoplasm, a thickened interstitium with infiltrates of interstitial macrophages and segmental leukocytes; a prominent feature was alveolar macrophages with ragged membranes. Three weeks after exposure, the interstitial response was largely resolved. In mice, the proliferative response was mainly found in the alveolar macrophage population and the interstitial and endothelial cells. Histopathological changes were similar to those found in rats, although less severe.

5/AU,TI,SO,DE,AB/16 (Item 16 from file: 154)
AU- Davis JK; Thorp RB; Parker RF; White H; Dziedzic D; D'Arcy J; Cassell GH
TI- Development of an aerosol model of murine respiratory mycoplasmosis in mice.
SO- <JN> Infect Immun
SO- <VO> 54 (1) p194-201
SO- <PY> Oct 1986

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DE- <MINOR> Aerosols; Disease Models, Animal; Mice; Mice, Inbred C3H; Mice, Inbred C57BL; Tissue Distribution

DE- <MAJOR> *Mycoplasma Infections --Microbiology --MI; *Respiratory Tract Infections --Microbiology --MI

AB- Animal models of murine respiratory mycoplasmosis due to Mycoplasma pulmonis provide excellent opportunities to study respiratory disease due to an infectious agent. The purpose of the present study was to develop and characterize an aerosol model for the production of murine respiratory mycoplasmosis in mice. The exposure of mice for 30 min to aerosols generated with a DeVilbiss 45 nebulizer in a nose-only inhalation chamber consistently reproduced typical lesions. The chamber was operated with a nebulizer air flow of 5.3 liters/min at 5.0 lb/in and a diluting air flow of 20 liters/min, with the nebulizer containing 5 ml of a suspension of viable M. pulmonis organisms (a concentration between 6 X 10(5) to 6 X 10(10) CFU/ml). Infective aerosol particles of less than a 4.0-micron median aerodynamic diameter with a geometric standard deviation of approximately 2.0 reached the lungs and were evenly distributed among the different lung lobes. A minimum 1.5-log loss of viability in the M. pulmonis suspension was demonstrated. With the exception of the 50% lethal dose, all of the parameters previously established by intranasal inoculation could be examined with the aerosol model. The major advantages of the aerosol model were excellent reproducibility of exposure (both between different experiments and between animals in a given experiment), the avoidance of anesthetization, and the ability to immediately deposit the majority of the organisms in the lung. The only disadvantage was the requirement for large volumes of mycoplasmal cultures.

5/AU,TI,SO,DE,AB/17 (Item 17 from file: 154)

AU- Bond JA; Ayres PH; Medinsky MA; Cheng YS; Hirshfield*D; McClellan RO

TI- Disposition and metabolism of [14C]dibenzo[c,g]carbazole aerosols in rats after inhalation.

SO- <JN> Fundam Appl Toxicol

SO- <VO> 7 (1) p76-85

SO- <PY> Jul 1986

DE- <MINOR> Aerosols; Carbazoles --Administration and Dosage --AD Carbazoles --Toxicity --TO Carbazoles --Urine --UR; Chromatography, High Pressure Liquid; Feces --Analysis --AN; Half-Life; Rats; Rats, Inbred Strains; Respiration --Drug Effects --DE; Respiratory System --Metabolism --ME; Time Factors; Tissue Distribution

DE- <MAJOR> *Carbazoles --Metabolism --ME

AB- Dibenzo[c,g]carbazole (DBC) is a nitrogen-containing polycyclic aromatic hydrocarbon that has been detected in tobacco tars, industrial oils, and diesel engine exhaust fumes. DBC is carcinogenic in respiratory tract tissue of hamsters and in lungs, kidneys, and livers of mice. The purpose of this research was to determine the respiratory tract deposition, distribution in tissues, metabolism, and excretion of DBC in rats after inhalation. Rats were exposed nose-only to 1.1 or 13 micrograms [14C]DBC/liter air for 60 min. Activity median aerodynamic diameters for the two concentrations of DBC ranged from 0.7 to 0.8 micron. Urine, feces, and selected tissues were collected for various times after exposure. The fractional deposition for the 1.1 and 13 micrograms/liter exposure concentrations was similar, 13 and 16%, respectively. The dominant route of excretion of 14C following exposure to either concentration of DBC was the feces, accounting for approximately 95% of the total 14C eliminated. Half-time for fecal excretion was 20 +/- 6 hr (means +/- SE). Gastrointestinal absorption of [14C]DBC was 43%. Radioactivity was widely distributed to all tissues examined, with the respiratory tract (lung, trachea, larynx, and nasal turbinates), upper gastrointestinal tract (stomach and small

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intestine), the liver, and the adrenals containing the highest concentrations of [14C]DBC equivalents within 1 hr after exposure. At both concentrations of DBC tested, clearance of 14C from tissues was rapid, with approximately 60 to 98% of the initial tissue burden being cleared with half-times ranging from 1 to 16 hr. The remaining 2 to 40% in the tissues was cleared with half-times that ranged from 1.5 to 14 days. Several metabolites were detected in the urine and feces, none of which appeared to be either glucuronide or sulfate conjugates. Small quantities of [14C]DBC were detected in the urine, although quantities were less than 1% of the initial respiratory tract burden of [14C]DBC. The results from this research indicate that DBC was rapidly absorbed from the lungs and translocated to many tissues. Prior to elimination, primarily in the feces, DBC was extensively metabolized. There appeared to be no effect of exposure concentration on the toxicokinetics of inhaled DBC.

5/AU,TI,SO,DE,AB/18 (Item 18 from file: 154)
AU- Bond JA; Dahl AR; Henderson RF; Dutcher JS; Mauderly JL; Birnbaum LS
TI- Species differences in the disposition of inhaled butadiene.
SO- <JN> Toxicol Appl Pharmacol
SO- <VO> 84 (3) p617-27
SO- <PY> Jul 1986
DE- <MINOR> Absorption; Atmosphere Exposure Chambers; Butadienes --Pharmacology --PD Butadienes --Urine --UR; Half-Life; Mice; Rats; Rats, Inbred Strains; Respiration --Drug Effects --DE; Species Specificity
DE- <MAJOR> *Butadienes --Metabolism --ME
AB- Recent chronic inhalation carcinogenicity studies of butadiene indicated that B6C3F1 mice are more sensitive, to the tumorigenic effects of inhaled butadiene than are Sprague-Dawley rats. Tumors in mice included lymphomas, hemangiosarcomas, alveolar/bronchiolar adenomas and carcinomas, and hepatocellular adenomas and carcinomas whereas in rats tumors included mammary tumors, thyroid follicular cell adenomas, uterine tumors, and exocrine pancreatic adenomas. The purpose of this investigation was to determine if there were differences in the uptake and disposition of inhaled butadiene between rats and mice and if these differences were consistent with the differences in the species susceptibility to inhaled butadiene. Male Sprague-Dawley rats and B6C3F1 mice were exposed nose only to concentrations in the range of 0.14 to 13,000 micrograms [14C]butadiene/liter air (0.08 to 7100 ppm; 25 degrees C, 620 torr) for 6 hr. Blood samples were taken during exposure and urine, feces, and expired air were collected for up to 65 hr after exposure. In both rats and mice there was a significant (p less than 0.001) concentration-related decrease in the percentage of butadiene retained at the cessation of a 6-hr exposure with increasing butadiene exposure concentration, suggesting saturable metabolism of this chemical. At all concentrations of butadiene tested, mice retained about 4 to 7 times the amount (mumol/kg body wt) of butadiene and metabolites than did rats. In both species and at all butadiene concentrations tested, urine and exhaled air were the major routes of excretion of 14C, together accounting for 75 to 85% of the total 14C eliminated. In mice, for all concentrations tested, elimination of 14C in urine, feces, and exhaled air increased with increasing butadiene exposure concentration, although the increase was not proportional to exposure concentration. However, exposure of rats to 13,000 micrograms butadiene/liter air resulted in a leveling off in the amount of 14C that was eliminated in urine and a concomitant increase in exhalation of 14CO2. Analysis of blood samples taken during exposure indicated that the blood of mice contained 2 to 5 times the concentration of 1,2-epoxy-3-butene than did the blood of rats. The data from this study indicate that species differences exist in the amount retained and

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metabolism of inhaled butadiene.

5/AU,TI,SO,DE,AB/19 (Item 19 from file: 154)
AU- Gairola CG
TI- Free lung cell response of mice and rats to mainstream cigarette smoke exposure.
SO- <JN> Toxicol Appl Pharmacol
SO- <VO> 84 (3) p567-75
SO- <PY> Jul 1986
DE- <MINOR> Acetylglucosaminidase --Metabolism --ME; Body Weight; Carboxyhemoglobin --Analysis --AN; Leucine Aminopeptidase --Metabolism --ME; Macrophages --Metabolism --ME; Mice; Mice, Inbred C57BL; Nucleotidases --Metabolism --ME; Pulmonary Alveoli --Enzymology --EN; Rats; Rats, Inbred F344; Species Specificity; Superoxide --Metabolism --ME; Tobacco
DE- <MAJOR> *Lung --Drug Effects --DE; *Smoke
AB- Male C57BL mice and F-344 rats were exposed through nose only to fresh mainstream smoke from one University of Kentucky Reference cigarette (2R1) daily under standardized conditions. At different exposure points, the lungs of room control (RM), sham control (SH), and smoke-exposed (SM) animals were lavaged and the number, composition, and properties of bronchoalveolar lavage (BAL) cells were studied. Significantly elevated levels of blood COHb and pulmonary aryl hydrocarbon hydroxylase activity indicated effective inhalation of smoke by animals. The BAL cell analysis showed that cigarette smoke induced a five- to sevenfold increase in the number of BAL cells in mice following 10- to 12-week exposure. The proportion of neutrophils (PMN) increased to about 18 +/- 3% in SM mice as compared to less than 1% in controls. Cessation of smoke treatments returned the PMN levels to those of controls within 5 weeks. Unlike mice, smoke exposure for up to 32 weeks failed to induce appreciable changes in the number and proportion of macrophages and neutrophils in rats. Large brown macrophages were observed in SM groups of both species. Functional analysis demonstrated that the BAL cells from SM mice but not rats released greater amounts of superoxides than controls under resting and phagocytically stimulated conditions. Enzymatic analysis of macrophages showed that the activity of N-acetylglucosaminidase was increased in SM groups of both species. The activity of 5'nucleotidase was significantly reduced in macrophages from SM mice but not rats. Activity of leucine aminopeptidase remained unaltered in both species. These results demonstrate distinct differences in the response of mice and rats to identically generated cigarette smoke.

5/AU,TI,SO,DE,AB/20 (Item 20 from file: 154)
AU- Henry CJ; Kouri RE
TI- Chronic inhalation studies in mice. II. Effects of long-term exposure to 2R1 cigarette smoke on (C57BL/Cum x C3H/AnfCum)F1 mice.
SO- <JN> J Natl Cancer Inst
SO- <VO> 77 (1) p203-12
SO- <PY> Jul 1986
DE- <MINOR> Adenocarcinoma --Etiology --ET; Adenoma --Etiology --ET; Body Weight; Bronchial Neoplasms --Etiology --ET; Carboxyhemoglobin --Analysis --AN; Data Collection; Disease Models, Animal; Fibrosarcoma --Etiology --ET; Head and Neck Neoplasms --Etiology --ET; Lung --Pathology --PA; Lung Neoplasms --Etiology --ET; Mice; Mice, Inbred Strains; Research Design; Respiration; Risk; Time Factors
DE- <MAJOR> *Smoke --Adverse Effects --AE; *Tobacco
AB- Standardized exposure conditions with Kentucky reference 2R1 cigarettes were used to expose 2,053 (C57BL/Cum x C3H/AnfCum)F1 female mice (nose only) to fresh, whole cigarette smoke. In addition, 1,014 mice were sham-exposed, and 449 mice were held as shelf controls. The protocol

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entailed exposing mice to smoke (or sham-exposure) on a daily basis, 5 days/week, for 110 weeks and observing remaining mice until death. A large number of animals was used so that the smoke generation and animal-holding systems could be tested and evaluated and yet provide significant numbers of animals for exposure to cigarette smoke for a major portion of their lifetime. Deposition of smoke particulates was estimated to be about 125-200 micrograms total particulate matter/lung/day. The only lung cancers observed were diagnosed as alveolar adenocarcinomas (AAC). A total of 19 of 978 smoke-exposed mice and 7 of 651 sham-exposed mice were observed with AAC. The difference between the smoke-and sham-exposed groups was not statistically significant at P less than or equal to .05, but the data suggested that the tumors occurred with a shorter latency in the smoke-exposed group (P = .10). The data were analyzed by various methods, including analysis of subsets of the population of animals. A significant increase in the incidence of lung cancer was observed in one subset; however, this difference was not found in the population as a whole or as a result of any other analyses. Under these exposure conditions, 2R1 cigarette smoke would seem to have weak carcinogenic activity in mouse lung tissue. Other changes associated with smoke exposure were increased incidence of pigmented alveolar macrophage accumulation, otitis media, and head and neck fibrosarcomas. However, the incidence of nephritis, hematopoietic cancers (e.g., leukemias, lymphosarcomas, and reticulum cell sarcomas), and pulmonary congestion was significantly higher in the sham-exposed animals.

5/AU,TI,SO,DE,AB/21 (Item 21 from file: 154)
AU- Thurman JD; Creasia DA; Quance JL; Johnson AJ
TI- Adrenal cortical necrosis caused by T-2 mycotoxicosis in female, but not male, mice.
SO- <JN> Am J Vet Res
SO- <VO> 47 (5) p1122-4
SO- <PY> May 1986
DE- <MINOR> Administration, Intranasal; Adrenal Cortex Diseases --Pathology --PA; Mice; Mice, Inbred ICR; Mycotoxins --Administration and Dosage --AD; Necrosis; Sex Factors; Thymus Gland --Pathology --PA
DE- <MAJOR> *Adrenal Cortex Diseases --Chemically Induced --CI; *Mycotoxins --Toxicity --TO
AB- Experimentally, adrenal cortical parenchymal cell necrosis was induced by T-2 mycotoxin in female, but not male, mice. The lesion occurred in the adrenal glands in 11 of 11 female and 0 of 10 male mice given a nose-only aerosol exposure to T-2 mycotoxin. The necrosis, restricted to the zona fasciculata, began at the X zone interface and extended peripherally to involve 15% to 70% of the zone. Both light and transmission electron microscopies were used to determine whether the cellular and subcellular damage involved parenchymal cells of the zona fasciculata. Extensive necrosis of cortical thymocytes and necrosis of lymphoid cells in follicles of the spleen, lymph nodes, and intestine-associated lymphoid tissue were observed in both sexes. This is the first report to describe adrenal gland necrosis associated with exposure to T-2 mycotoxin.

5/AU,TI,SO,DE,AB/22 (Item 22 from file: 154)
AU- Philpott DE; Sapp W; Miquel J; Kato K; Corbett R; Stevenson J; Black S; Lindseth KA; Benton EV
TI- The effect of high energy (HZE) particle radiation (40Ar) on aging parameters of mouse hippocampus and retina.
SO- <JN> Scan Electron Microsc
SO- <VO> (Pt 3) p1177-82

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SO- <PY> 1985
DE- <MINOR> Argon --Administration and Dosage --AD; Body Weight --Radiation Effects --RE; Cell Count; Dose-Response Relationship, Radiation; Hippocampus --Physiology --PH; Mice; Mice, Inbred C57BL; Motor Activity --Radiation Effects --RE; Radiation Injuries, Experimental --Pathology --PA; Retina --Physiology --PH; Synapses --Radiation Effects --RE
DE- <MAJOR> *Aging --Radiation Effects --RE; *Hippocampus --Radiation Effects --RE; *Radiation Injuries, Experimental --Physiopathology --PP; *Retina --Radiation Effects --RE
AB- Eight month old C57BL6 mice were exposed (head only) to Ø.5 rad or 5Ø rads of Argon particles at the Lawrence Berkeley Radiation Facility, CA. Neuromotor performance was assessed monthly for six months beginning twelve weeks post-irradiation using a "string test". The decline in motor performance was dose-related and none of the animals was able to complete the task after four months of testing. Morphological changes were monitored six and twelve months post-irradiation by light and electron microscopy. The synaptic density in the CA-1 area of the hippocampus decreased six and twelve months after irradiation. The decrease after twelve months was less than after six months. The width of the outer nuclear layer (ONL) of the retina increased with increasing dose. The number of blood vessels between the ONL and the ganglion layer decreased twelve months after irradiation and this area did not show significant accumulation of age pigment.

5/AU,TI,SO,DE,AB/23 (Item 23 from file: 154)
AU- Graziano MJ; Gairola C; Dorough HW
TI- Effects of cigarette smoke and dietary vitamin E levels on selected lung and hepatic biotransformation enzymes in mice.
SO- <JN> Drug Nutr Interact
SO- <VO> 3 (4) p213-22
SO- <PY> 1985
DE- <MINOR> Aryl Hydrocarbon Hydroxylases --Metabolism --ME; Biotransformation; Body Weight; Caloric Intake; Diet; Glutathione --Metabolism --ME; Lipid Peroxides --Biosynthesis --BI; Mice; Mice, Inbred C57BL; Organ Weight; Proteins --Metabolism --ME
DE- <MAJOR> *Liver --Enzymology --EN; *Lung --Enzymology --EN; *Smoke --Adverse Effects --AE; *Tobacco; *Vitamin E Deficiency --Enzymology --EN
AB- Young male C57BL mice were exposed nose-only to cigarette smoke 2Ø min/day for 8 weeks while maintained on diets containing Ø, 5, and 1ØØ ppm of vitamin E. Smoking had no effect on hepatic aryl hydrocarbon hydroxylase (AHH), UDP-glucuronyltransferase, glutathione S-transferase, parathion desulfurase, or parathion esterase activity. Lung AHH activity was increased in all smoke-exposed mice, although the increase was significantly less in animals maintained on the vitamin E-free diet. All mice on the vitamin E-free diet showed reduced lung AHH activity and increased hepatic lipid peroxidation. No other biotransformations tested were significantly altered by varying vitamin E concentrations alone or in combination with cigarette smoke. For all vitamin E diets, both the smoke-exposed and sham-treated mice gained significantly less weight than the control animals. This effect was attributed to stress induced by restraint of the animals within the smoking apparatus. The results of these experiments show that both cigarette smoke and vitamin E-deficient diets may affect xenobiotic metabolism but that the combination does not appear to alter markedly their individual effects or to induce ones not previously observed.

5/AU,TI,SO,DE,AB/24 (Item 24 from file: 154)
AU- Babiuk C; Steinhagen WH; Barrow CS
TI- Sensory irritation response to inhaled aldehydes after formaldehyde pretreatment.

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SO- <JN> Toxicol Appl Pharmacol
SO- <VO> 79 (1) p143-9
SO- <PY> Jun 15 1985
DE- <MINOR> Aldehydes --Administration and Dosage --AD; Atmosphere Exposure Chambers; Dose-Response Relationship, Drug; Drug Tolerance; Formaldehyde --Administration and Dosage --AD; Irritants --Administration and Dosage --AD; Mice; Rats; Rats, Inbred F344; Respiration --Drug Effects --DE; Species Specificity; Time Factors
DE- <MAJOR> *Aldehydes --Toxicity --TO; *Formaldehyde --Toxicity --TO; *Irritants
AB- Pretreatment of Fischer-344 (F-344) rats with formaldehyde (HCHO) induces significant cross tolerance to the sensory irritation properties of C12. The purpose of this study was to determine if HCHO pretreatment would cause sensory irritation cross tolerance to other inhaled aldehydes. Male F-344 rats, weighing 190 to 210 g, were pretreated with 15 ppm HCHO, 6 hr/day for 9 days, and challenged on the 10th day with a saturated (acetaldehyde, propionaldehyde, and butyraldehyde), unsaturated (acrolein and crotonaldehyde), or cyclic (cyclohexanecarboxaldehyde, 3-cyclohexene-1-carboxaldehyde, and benzaldehyde) aldehyde. The sensory irritation response in these animals was quantified by measuring respiratory rate depression in a head-only inhalation chamber using plethysmographic techniques. Control animals were challenged identically without prior pretreatment. In naive (nonpretreated) animals, the concentration eliciting a 50% decrease in respiratory rate (RD50) was 23 ppm or less for unsaturated aliphatic aldehydes. For cyclic and saturated aliphatic aldehydes, the RD50 ranged from 600 to 1000 ppm and 3000 to 6800 ppm, respectively. Formaldehyde pretreatment resulted in cross tolerance only with acetaldehyde (RD50 increased 3.5-fold) and acrolein (RD50 increased 5-fold). These results indicate that the development of cross tolerance following HCHO pretreatment is not a general phenomenon. Prediction of acceptable concentrations of occupational exposure for the prevention of sensory irritation in humans has been based primarily on RD50 data for mice. Comparison of the RD50 values obtained for rats in this investigation with previously published results for mice varied by over one-half an order of magnitude, thereby disputing the usefulness of data from F-344 rats in setting threshold limit values for the prevention of sensory irritation.

5/AU,TI,SO,DE,AB/25 (Item 25 from file: 154)
AU- Putman DL; David RM; Melhorn JM; Dansie DR; Stone CJ; Henry CJ
TI- Dose-responsive increase in sister-chromatid exchanges in bone-marrow cells of mice exposed nose-only to whole cigarette smoke.
SO- <JN> Mutat Res
SO- <VO> 156 (3) p181-6
SO- <PY> Jun 1985
DE- <MINOR> Bone Marrow --Cytology --CY; Mice; Nose; Time Factors; Tobacco
DE- <MAJOR> *Mutation; *Sister Chromatid Exchange; *Smoke
AB- The effect of whole cigarette smoke exposure on bone-marrow sister-chromatid exchanges (SCEs) was studied in B6C3F1 mice. Animals were exposed nose-only to 10% (v/v) cigarette smoke 5 days/week for 2 weeks. Four dose levels of cigarette smoke (1, 4, 9 and 18 exposures/day) were studied using 2 cigarette types, Kentucky reference 3A1 (3A1) and American Blend (AB). A single exposure represented approximately 1 cigarette. A dose-dependent increase in SCEs was observed for both the 3A1 and AB cigarettes at dose levels which had no effect on bone-marrow cell-replication kinetics. These findings represent the first demonstration of a dose-responsive increase in cigarette smoke-induced SCEs in a rodent model system.

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5/AU,TI,SO,DE,AB/26 (Item 1 from file: 55)

AU- MCANULTY R J; MOORES S R; TALBOT R J; BISHOP J E; MAYS P K; LAURENT G J

TI- LONG-TERM CHANGES IN MOUSE LUNG FOLLOWING INHALATION OF A FIBROSIS-INDUCING DOSE OF PLUTONIUM-239 OXIDE CHANGES IN COLLAGEN SYNTHESIS AND DEGRADATION RATES

SO- <JN> International Journal of Radiation Biology

SO- <JN> INT J RADIAT BIOL

SO- <PY> 59 (1). 1991. 229-238.

DE- CONNECTIVE TISSUE MATRIX REMODELLING HYDROXYPROLINE THORACIC RADIOTHERAPY COMPLICATION TUMOR

AB- Mice were exposed by nose-only inhalation to 239PuO2, which resulted in an IAD of 1110.+-.29 Bq. At various times after exposure, rates of collagen metabolism were measured using validated in vivo methods based on the administration of radiolabelled proline, together with a large flooding dose of unlabelled proline and measurements of its incorporation into lung collagen as hydroxyproline. Dramatic increases in both synthesis and degradation rates of collagen were observed. At 54 days after exposure the fractional synthesis rates in experimental mice were almost five times those in controls (around: 3.2.+-.0.6%/day, 239PuO2-exposed: 14.5.+-.0.4%/day) and by 300 days synthesis rates, although declining, were still more than double the control values. A similar pattern of change was observed for collagen degradation. The combination of changes in synthesis and degradation rates led to a 60% increase in lung collagen content by 300 days (control: 3.05.+-.0.24 mg/lung, 239PuO2-exposed: 4.88.+-.0.42 mg/lung). The data suggest that extensive remodeling of the lung connective tissue matrix occurs during development of fibrosis and that, over long periods of time, small imbalances between synthesis and degradation may result in quite large increases in protein content.

5/AU,TI,SO,DE,AB/27 (Item 2 from file: 55)

AU- ISHIDA M; MIZUHASHI T; MURASE H; KAMEDA M; TSUJI K; MURAKAMI Y; OHTA Y; KUROSAKI T; WAKI I

TI- SINGLE AND REPEATED DOSE TOXICITY OF PERCUTANEOUSLY APPLIED CADEXOMER IN RODENTS

SO- <JN> Pharmacometrics

SO- <JN> PHARMACOMETRICS

SO- <PY> 39 (5). 1990. 473-484.

DE- DERMATOLOGICAL-DRUG DECREASED LYMPHOCYTE LEVEL CHOLESTEROL LEVEL ADRENAL WEIGHT THYMUS WEIGHT SKIN ULCER

AB- Cadexomer is presented as polymerized granules of dextrin and used as the base of NI-009, a new powder drug for external application to skin ulcer. A study was made of the local and systemic effects of single and repeated occlusive application of cadexomer with a dressing to the stratum corneum-removed skin of rodents. Single dose toxicity was determined in ddY mice and Wistar rats. Throughout the experimental period, there occurred no deaths, no skin reactions, no toxic signs. Percutaneous LD50 values were thus more than 10 g/kg in mice and rats of both sexes. Repeated dose toxicity was determined in male and female Wistar rats; cadexomer powder was given percutaneously at dose levels of 0 (procedural control), 0.2, 0.4 or 0.8 g/head/day for 30 consecutive days. Cadexomer powder caused no death, produced no change in the skin and had no gross behavioral effect at any dose level throughout the experimental period. Nor did it have any significant effect on body weight or food consumption, except a transient decrease of water intake in females at 0.4 and 0.8 g/head. Only at 0.2 g/head cadexomer caused several significant changes in males. The changes included a decrease in the percentage of lymphocytes in the blood, an increase in total serum cholesterol, an increase in adrenal weight and

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a decrease in thymus weight. There were no marked changes related to cadexomer at Ø.4 or Ø.8 g/head. Accordingly, none of the changes were dose-related. The changes seen were probably due to the stress presented by the dressing. Therefore, it is concluded that the maximum no-effect dose level is more than Ø.8 g/head/day for male and female rats.

5/AU,TI,SO,DE,AB/28 (Item 3 from file: 55)
AU- KUNA P
TI- RADIOPROTECTIVE EFFECT OF WR-2721 GAMMAPHOS OR CYSTAMINE IN NON-UNIFORM LETHAL GAMMA IRRADIATED MICE
SO- <JN> Radiobiologia Radiotherapia
SO- <JN> RADIOBIOL RADIOTHER
SO- <PY> 27 (6). 1986 (RECD. 1987). 761-77Ø.
DE- RADIOPROTECTORANT-DRUG
AB- The radioprotective effects of intramuscularly administered WR-2721 (3ØØ mg .cntdot. kg-1) or cystamine (15Ø mg .cntdot. kf-1) were studied in cephalic or extracephalic gamma irradiated mice. Lead shielding of the head of the mouse gives DRFs equal to 2.32-2.53. The DRF values were determined on 3.24-3.78 in mice when head only was irradiated and the remainder of the body was protected by lead layer. An addition of chemical protectors to physical shield increased antiradiation effect. Cystamine was insignificantly better than WR-2721 in protection of head irradiated mice. WR-2721 was more effective than cystamine in combination with the head shielding of the mice, similarly as in the case of whole body gamma irradiated mice.

5/AU,TI,SO,DE,AB/29 (Item 4 from file: 55)
AU- TYL R W; BALLANTYNE B; FAIT D L; FISHER L C; SAVINE T A; PRITTS I M; DODD D E

TI- TERATOGENICITY EVALUATION OF WATER AEROSOL AER VERSUS AIR AIR IN CD-1 MICE BY WHOLE BODY WB OR NOSE-ONLY NO EXPOSURE
SO- <JN> TWENTY-SEVENTH ANNUAL MEETING OF THE TERATOLOGY SOCIETY, RANCHO MIRAGE, CALIFORNIA, USA, JUNE 14-18, 1987. TERATOLOGY
SO- <PY> 35 (2). 1987. 39A.
DE- ABSTRACT INDUSTRIAL CHEMICAL EXPOSURE

5/AU,TI,SO,DE,AB/3Ø (Item 5 from file: 55)
AU- HENRY C J; KOURI R E
TI- CHRONIC INHALATION STUDIES IN MICE II. EFFECTS OF LONG-TERM EXPOSURE TO 2R-1 CIGARETTE SMOKE ON C-57BL-CUM X C-3H-ANFCUM F-1 MICE
SO- <JN> Journal of the National Cancer Institute
SO- <JN> J NATL CANCER INST
SO- <PY> 77 (1). 1986. 2Ø3-212.
DE- WEAK CARCINOGEN NASAL ADMINISTRATION SMOKE PARTICLE DEPOSIT ALVEOLAR ADENOCARCINOMA PIGMENTED ALVEOLAR MACROPHAGE ACCUMULATION OTITIS MEDIA HEAD FIBROSARCOMA NECK FIBROSARCOMA NEPHRITIS HEMATOPOIETIC CANCER PULMONARY CONGESTION
AB- Standardized exposure conditions with Kentucky reference 2R1 cigarettes were used to expose, 2,Ø53 (C57BL/Cum .times. C3H/AnfCum)F1 female mice (nose only) to fresh, whole cigarette smoke. In addition, 1,Ø14 mice were sham-exposed, and 449 mice were held as shelf controls. The protocol, entailed exposing mice to smoke (or sham-exposure) on a daily basis, 5 days/week, for 11Ø weeks and observing remaining mice until death. A large number of animals was used so that the smoke generation and animal-holding systems could be tested and evaluated and yet provide significant numbers of animals for exposure to cigarette smoke for a major portion of their lifetime. Deposition of smoke particulates was estimated to be about 125-2ØØ .mu.g total particulate

Source: https://www.industrydocuments.ucsf.edu/docs/gndl0000

matter/lung/day. The only lung cancers observed were diagnosed as aleveolar adenocarcinomas (AAC). A total of 19 of 978 smoke-exposed mice and 7 of 651 sham-exposed mice were observed with AAC. The difference between the smoke- and sham-exposed groups was not statistically significant at P .gtoreq. .05, but the data suggested that the tumors occurred with a shorter latency in the smoke-exposed group (P = .10). The data were analyzed by various methods, including analysis of subsets of the population of animals. A significant increase in the incidence of lung cancer was observed in one subset; however, this difference was not found in the population as a whole or as a result of any other analyses. Under these exposure conditions, 2R1 cigarette smoke would seem to have weak carcinogenic activity in mouse lung tissue. Other changes associated with smoke exposure were increased incidence of pigmented alveolar macrophage accumulation, otitis media, and head and neck fibrosarcomas. However, the incidence of nephritis, hematopoietic cancers (e.g., leukemias, lymphosarcomas, and reticulum cell sarcomas), and pulmonary congestion was significantly higher in the sham-exposed animals.

5/AU,TI,SO,DE,AB/31 (Item 6 from file: 55)

AU- BOND J A; AYRES P H; MEDINSKY M A; CHENG Y-S; HIRSHFIELD D; MCCLELLAN R O

TI- DISPOSITION AND METABOLISM OF CARBON-14 DIBENZO-C G-CARBAZOLE AEROSOLS IN RATS AFTER INHALATION

SO- <JN> Fundamental and Applied Toxicology

SO- <JN> FUNDAM APPL TOXICOL

SO- <PY> 7 (1). 1986.. 76-85.

DE- MOUSE HAMSTER LUNG KIDNEY LIVER UPPER GASTROINTESTINAL TRACT CARCINOGEN TOXICOKINETICS

AB- Dibenzo[c,g]carbazole (DBC) is a nitrogen-containing polycyclic aromatic hydrocarbon that has been detected in tobacco tars, industrial oils, and diesel engine exhaust fumes. DBC is carcinogenic in respiratory tract tissue of hamsters and in lungs, kidneys, and livers of mice. The purpose of this research was to determine the respiratory tract deposition, distribution in tissues, metabolism, and excretion of DBC in rats after inhalation. Rats were exposed nose-only to 1.1 or 13 .mu.g [14C]DBC/liter air for 60 min. Activity median aerodynamic diameters for the two concentrations of DBC ranged from 0.7 to 0.8 .mu.m. Urine, feces, and selected tissues were collected for various times after exposure. The fractional deposition for the 1.1 and 13 .mu.g/liter exposure concentrations was similar, 13, and 16%, respectively. The dominant route of excretion of 14C following exposure to either concentration of DBC was the feces, accounting for approximately 95% of the total 14C eliminated. Half-time for fecal excretion was 20 .+-. 6 hr (.hivin.x .+-. SE). Gastrointestinal absorption of [14C]DBC was 43%. Radioactivity was widely distributed to all tissues examined, with the respiratory tract (lung, trachea, larynx, and nasal turbinates), upper gastrointestinal tract (stomach and small intestine), the liver, and the adrenals containing the highest concentrations of [14C]DBC equivalents within 1 hr after exposure. At both concentrations of DBC tested, clearance of 14C from tissues was rapid, with approximately 60 to 98% of the initial tissue burden being cleared with half-times ranging from 1 to 16 hr. The remaining 2 to 40% in the tissues was cleared with half-times that ranged from 1.5 to 14 days. Several metabolites were detected in the urine and feces, none of which appeared to be either glucuronide or sulfate conjugates. Small quantities of [14C]DBC were detected in the urine, although quantities were less than 1% of the initial respiratory tract burden of [14C]DBC. The results from this research indicate that DBC was rapidly absorbed from the lungs and translocated to many tissues. Prior to elimination, primarily in the feces, DBC was

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extensively metabolized. There appeared to be no effect of exposure concentration on the toxicokinetics of inhaled DBC.

5/AU,TI,SO,DE,AB/32 (Item 7 from file: 55)
AU- PHILPOTT D E; SAPP W; MIQUEL J; KATO K; CORBETT R; STEVENSON J; BLACK
S; LINDSETH K A; BENTON E V
TI- THE EFFECT OF HIGH ENERGY HZE HIGH ATOMIC NUMBER PARTICLES PARTICLE RADIATION ARGON-40 ON AGING PARAMETERS OF MOUSE HIPPOCAMPUS AND RETINA
SO- <JN> Scanning Electron Microscopy
SO- <JN> SCANNING ELECTRON MICROSC
SO- <PY> 1985 (3). 1985. 1177-1182.
DE- GANGLION LETTER OUTER NUCLEAR LAYER NEUROMOTOR PERFORMANCE
AB- Eight month old C57BL6 mice were exposed (head only) to 0.5 rad or 50 rads of Argon particles at the Lawrence Berkeley Radiation Facility, CA. Neuromotor performance was assessed monthly for six months beginning twelve weeks post-irradiation using a "string test". The decline in motor performance was dose-related and none of the animals was able to complete the task after four months of testing. Morphological changes were monitored six and twelve months post-irradiation by light and electron microscopy. The synaptic density in the CA-1 area of the hippocampus decreased six and twelve months after irradiation. The decrease after twelve months was less than after six months. The width of the outer nuclear layer (ONL) of the retina increased with increasing dose. The number of blood vessels between the ONL and the ganglion layer decreased twelve months after irradiation and this area did not show significant accumulation of age pigment.

5/AU,TI,SO,DE,AB/33 (Item 8 from file: 55)
AU- HENRY C J; DANSIE D R; KANAGALINGAM K K; KOURI R E; GAYLE T; GUERIN M; HOLMBERG R; FLORANT L; GREENSPAN J
TI- CHRONIC INHALATION STUDIES IN MICE 1. FACILITIES AND EQUIPMENT FOR NOSE-ONLY EXPOSURE TO CIGARETTE SMOKE
SO- <JN> Beitraege zur Tabakforschung International
SO- <JN> BEITR TABAKFORSCH INT
SO- <PY> 13 (1). 1985. 37-54.
DE- LUNG
AB- Facilities and equipment are described for large-scale, long-term nose-only inhalation exposure of mice to whole cigarette smoke. Experimental procedures and equipment were designed to provide the mice with exposure conditions where the lung was the major target organ for the smoke, large quantities of fresh, whole cigarette smoke could be generated, large numbers of animals could be exposed at one time, routine, daily exposures could be given over a major portion of the lifetime of the animal, monitoring and documentation of the quantity of smoke presented to the animals was provided during each exposure session, safety systems were provided that assured exposure of the animals to smoke only under pre-set exposure conditions and cigarette smoke was generated under conditions where factors, such as cigarette type, smoke aerosol concentration and smoke particle size, were controlled.

5/AU,TI,SO,DE,AB/34 (Item 9 from file: 55)
AU- STADLER J; KAROL M H
TI- EXPERIMENTAL DELAYED HYPERSENSITIVITY FOLLOWING INHALATION OF DICYCLOHEXYLMETHANE 4 4' DIISOCYANATE A CONCENTRATION-RESPONSE RELATIONSHIP
SO- <JN> Toxicology and Applied Pharmacology
SO- <JN> TOXICOL APPL PHARMACOL

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SO- <PY> 74 (2). 1984. 244-249.
DE- HUMAN WORKER MOUSE GUINEA-PIG CONTACT DERMATITIS
AB- Exposure of workers to low MW chemicals has been associated with delayed-onset hypersensitivity reactions in the skin and respiratory tract. The use of 2 animal models to examine factors affecting the production of delayed hypersensitivity to dicyclohexylmethane-4,4'-diisocyanate (HMDI) was described. Guinea pigs were exposed to HMDI by a head-only method and mice in a nose-only procedure. All animals were tested for dermal sensitivity by topical challenge with the isocyanate. In both species, a concentration-response relationship and no-effect concentration was observed between chamber concentration of HMDI and severity of the dermal response and number of animals responding. Guinea pigs developed skin sensitivity following inhalation of .gtoreq. 3 .mu.g HMDI for 2 h/day on 3 consecutive days. Inhalation of 1.25 .mu.g/l did not result in sensitization. Contact sensitivity was detected in BALB/cBy mice following inhalation of .gtoreq. 17 .mu.g/HMDI. No reactions occurred as a result of exposure to .ltoreq. 7 .mu.g/l HMDI. Dermal contact of the head, as would occur during inhalation exposures, also resulted in contact sensitization. Identification of the concentration-response relationship for contact sensitization following inhalation exposure implied that safe exposure levels can be proposed to prevent cases of dermal sensitization to HMDI.

5/AU,TI,SO,DE,AB/35 (Item 1 from file: 156)
TI- INITIAL SUBMISSION: ETHYLENE GLYCOL: DEVELOPMENTAL TOXICITY EVALUATION OF THE AEROSOL IN CD-1 MICE BY NOSE ONLY OR WHOLE BODY EXPOSURE (FINAL REPORT) W-COVER LETTER DATED 103191
SO- EPA/OTS; Doc 88-920000119
DE- UNION CARBIDE CORP; ETHYLENE GLYCOL; HEALTH EFFECTS; REPRODUCTION/FERTILITY EFFECTS; TERATOGENICITY; MAMMALS; MICE; INHALATION

5/AU,TI,SO,DE,AB/36 (Item 2 from file: 156)
AU- Hume AS
TI- Chemoprophylaxis and Antidotal Efficacy of Alpha-Ketoglutaric Acid in Hydrogen Cyanide Poisoning.
SO- Govt Reports Announcements & Index (GRA&I), Issue 08, 1992
DE- Antidotes, Cardiovascular system, Chemistry, Colorimetric analysis, Detectors, Determination, Electrochemistry, Hydrogen cyanide, Inhalation, Lethality, Lung, Mechanics, Mice, Models, Monitoring, Potency,; Preventive medicine, Rats, Toxicity; Cyanide antidotes, Inhalation chamber, Hypoxia; Cyanide poisoning
AB- TD3: A model to study the toxic effect for hydrogen cyanide (HCN) by inhalation and to evaluate the efficiencies and relative potencies of possible antidotes has been established. Several chambers and accompanying mechanics of administration of HCN were designed and evaluated. This model is especially designed to be used to study the effects of HCN at different concentrations and to evaluate the effectiveness of the antidotes with pre- and post-exposure of mice and rats to HCN. In addition, pulmonary and cardiovascular changes in the rat can be monitored. This allows more discrete determination of the efficacy of antidotes and monitoring of toxic effects other than lethality. During exposure, HCN concentrations in the chamber were continuously monitored by detector tubes, electrochemical sensors, and chemical colorimetric methods. Mice were placed in a cage which was quickly inserted and removed from the chamber. Rats were placed in a rat hold (tube) for insertion of nose-only exposure. Final rept. 14 Mar 88-13 Sep 90.

5/AU,TI,SO,DE,AB/37 (Item 3 from file: 156)

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TI- SUPPLEMENTAL INFORMATION: TERATOGENICITY EVALUATION OF WATER AEROSOL OR AIR IN CD-1 MICE BY NOSE ONLY OR WHOLE BODY EXPOSURE (PROJECT REPORT) WITH COVER LETTER DATED Ø82891
SO- EPA/OTS; Doc FYI-OTS-Ø991-Ø323
DE- UNION CARBIDE CORP; 1,2-ETHANEDIOL; HEALTH EFFECTS; REPRODUCTION/FERTILITY EFFECTS; TERATOGENICITY; MAMMALS; MICE; INHALATION

5/AU,TI,SO,DE,AB/38 (Item 4 from file: 156)
TI- ETHYLENE GLYCOL: DEVELOPMENTAL TOXICITY EVALUATION OF THE AEROSOL IN CD-1 MICE BY NOSE ONLY OR WHOLE BODY EXPOSURE WITH COVER LETTER DATED Ø42988
SO- EPA/OTS; Doc FYI-OTS-Ø588-Ø323
DE- UNION CARBIDE CORP; ETHYLENE GLYCOL; HEALTH EFFECTS; REPRODUCTION/FERTILITY EFFECTS; TERATOGENICITY; MAMMALS; MICE; INHALATION
AB- Teratogenicity was eval- uated in groups of 3Ø pregnant CD-1 albino mice dosed by inhalation 6 hrs/day gestation days (GD) 6-15 at total ethylene glycol concentrations generated as an aerosol and measured by gas chromatography at (nose-only, restrained) Ø (water), 25Ø-36Ø, 57Ø-779, or 969-25Ø5 mg/m3, aerosol of particle size 1.Ø um MMAD +/- 4.2 GSD, or (whole-body, unrestrained, positive control) Ø (water) or 2ØØ8 mg/m3, aerosol of particle size 7.5 um MMAD +/- 1.6 GSD. Groups of 5 dams either nose- or whole-body exposed to top doses GD 6 showed whole-body exposed dams had >1ØØØ more mg/kg extractable ethylene glycol deposited on fur. Nose- exposed maternal mortalities (2 and 6 in Ø and high dose groups, respectively) were believed to be dosing errors. Early deliveries (total resorptions) for negative control, low, mid, high, and whole body dose groups were 3, 3, (2), (1), 1, and 1. Maternal signs of clinical toxicity (other than restraint-in- duced in nose-only groups) were wet fur in both high dose groups. Treatment increased water consumption in low dose and whole-body dams. Dam weights and weight gains were unaffected. Absolute (mid and high dose) and relative (high dose) dam kidney weights increased. The number of corpora lutea/dam, total or viable implants/litter, and sex ratio were unaffected. Nonviable implants/ litter increased in whole-body treated group. Fetal weights were reduced in both high-dose groups. There was no treatment-related effect on soft tissue anomalies in fetuses, but skeletal effects were seen both high dose groups.

5/AU,TI,SO,DE,AB/39 (Item 5 from file: 156)
AU- Carchman RA
TI- Characterization of Nerve Agent Exposure on Alveolar Macrophage Function.
SO- Govt Reports Announcements & Index (GRA&I), Issue Ø1, 1989
DE- Alveoli, Anions, Assaying, Base lines, Calcium, Cells, Chambers, Chemotaxis, Exposure(Physiology), Functions, Guinea pigs, High rate, Inhalation,; Nerve agents; Phagocytes, Response(Biology), Reticuloendothelial system, Stimulation(Physiology), Superoxides, Targets, Physiological effects
AB- TD3: Guinea pig alveolar cells (GPAM) were obtained in situ via bronchoalveolar lavage. The following functional assays were studied in the GPAM: Chemotaxis. 1) Chemotaxis which was stimulated by N-formyl-methionyl-leucine-phenylalanine (FMLP) and phorbol-12-myristate-13-acetate (PMA) in a concentration-related manner. 2) Cytotoxicity as measured by 51Cr release from target cells + or - PMA was induced in P815 mastocytoma cells and less strongly in 3T3 normal mouse fibroblasts. 3) Release of N-acetyl-Beta-D-glucosaminidase (NAGA) was stimulated by the calcium ionophore, A23187, but not by PMA or the combination of A23187+PMA. 4) Phagocytosis of opsonized 51Cr sheep red blood cells occurred in a time-related manner and reached its maximum after 12Ø minutes. 5) Superoxide anion production as measured by the

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reduction of ferricytochrome C was stimulated 25 fold by PMA. 6) Cell spreading, which exhibited a high rate of spontaneous spreading was only minimally stimulatable by PMA. In addition to establishing the baseline response for naive GPAM, the efficiency of the inhalation chamber was maximized to allow for the volatilization and 'nose-only' delivery of organophosphates. (KT) Annual rept. 1 Jul 85-30 Jun 86.

5/AU,TI,SO,DE,AB/40 (Item 6 from file: 156)
AU- TYL RW; BALLANTYNE B; FAIT DL; FISHER LC; SAVINE TA; PRITTS IM; DODD DE
TI- TERATOGENICITY EVALUATION OF WATER AEROSOL VERSUS AIR IN CD-1 MICE BY WHOLE BODY OR NOSE-ONLY EXPOSURE
SO- TERATOLOGY 35(2):39A,1987
DE- MUS, CD1; MAMMAL, MOUSE; FEMALE; VIABILITY, FERTILITY AND MORTALITY; NERVOUS SYSTEM; SENSE ORGANS; CRANIUM AND FACE; CARDIOVASCULAR SYSTEM; MUSCULOSKELETAL SYSTEM; GROWTH; MATERNAL WEIGHT CHANGES; MATERNAL LIVER; FETAL LOSS; INHALATION; AEROSOLS; WATER

5/AU,TI,SO,DE,AB/41 (Item 7 from file: 156)
AU- Raabe OG; Al-Bavati MA; Teague SV; Rasolt A
TI- Regional Deposition of Inhaled Monodisperse Coarse and Fine Aerosol Particles in Small Laboratory Animals.
SO- Govt Reports Announcements & Index (GRA&I), Issue 04, 1986
DE- Aerosols; Toxicology; Particulates; Air pollution effects(Animals)
AB- TD3: The regional deposition of inhaled monodisperse insoluble particles of about 1, 3, 5, and 10 micrometers in activity median aerodynamic diameter was measured in four small rodent species including CF1 mice, golden Syrian hamsters, Fischer 344 rats and Hartley guinea pigs and in New Zealand rabbits. Near monodisperse aerosols of about 0.05 micrometers activity median diffusive diameter were also studied. The five species are commonly utilized in inhalation toxicology research. Monodisperse aerosols of fused aluminosilicate particles labeled with radioactive 169Yb were generated utilizing a modified vibrating liquid stream generator, reduced to Boltzmann charge equilibrium with a 85Kr discharge device, concentrated with a centripeter stage, fused in a 1200C quartz tube furnace, and delivered to a specially designed nose-only exposure system suitable for use with coarse particles as large as 10 micrometers. Twenty unanaesthetized animals (eight rabbits) were exposed simultaneously for up to 45 minutes to each aerosol particle size. Sponsored by Health Effects Research Lab., Research Triangle Park, NC.

5/AU,TI,SO,DE,AB/42 (Item 8 from file: 156)
AU- WOLFF RK; KANAPILLY GM; GRAY RH; MCCLELLAN RO
TI- Deposition and retention of inhaled aggregate gallium-67 oxide particles in Beagle dogs, Fischer 344 rats and CD-1 mice.
SO- AM IND HYG ASSOC J; 45 (6). 1984. 377-381.
AB- HEEP COPYRIGHT: BIOL ABS.
AB- Deposition and retention of inhaled 67Ga2O3 aerosols were measured in 10 Beagle dogs to provide reasonable estimates for human deposition and in Fischer-344 rats and CD-1 mice to estimate lung burdens in small animals frequently used in toxicological evaluations. Aggregated particles of 67Ga2O3, 0.1 mum mass median diameter (MMD), were produced using heat treatment of 67Ga tetramethylheptanedione. Whole-body counting and gamma-camera imaging were used to measure deposition. Pulmonary deposition in dogs was measured as 25% of the inhaled particles for the 0.1 mum particles. Tracheobronchial and nasopharyngeal deposition were much lower, 7 and 7%, respectively. Pulmonary deposition of 0.1 mum (MMD) particles was calculated as 10 and 11% of inhaled particles in Fischer-344 rats and 15 and 20% in mice for 2 separate nose-only exposures.

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5/AU,TI,SO,DE,AB/43 (Item 9 from file: 156)
AU- BENEDICT WF; BANERJEE A; KANGALINGAM KK; DANSIE DR; KOURI RE; HENRY CJ
TI- Increased sister chromatid exchange in bone marrow cells of mice exposed to whole cigarette smoke.
SO- MUTAT RES; 136 (1). 1984. 73-80.
AB- HEEP COPYRIGHT: BIOL ABS.
AB- Using defined cigarette smoke exposure conditions, BC3F1/Cum mice were exposed nose-only to 2 different types of whole cigarette smoke on a daily basis for 1 wk and up to 46 wk. The number of sister-chromatid exchanges (SCE) per metaphase was determined in bone-marrow cells. Studies were scheduled so that all cytogenetic observations were made 2-3 days after the last smoke exposure. Exposure to either type of smoke on a daily basis for 1 wk or up to 46 wk resulted in a 2-fold increase in SCE over sham-exposed control mice. In animals exposed either chronically or for 1 wk to either type of smoke, the increase in SCE persisted for at least 1 wk after cessation of smoke exposure. This is the first demonstration of the induction of SCE in laboratory animals that were exposed to cigarette smoke in vivo.

5/AU,TI,SO,DE,AB/44 (Item 10 from file: 156)
AU- HENRY CJ; HWANG KK; KANAGALINGAM KK; KOURI RE
TI- RECENT DEVELOPMENTS IN INHALATION TOXICOLOGY EVALUATION OF SELECTED SHORT-TERM END POINTS FOLLOWING NOSE ONLY EXPOSURE OF RODENTS
SO- HOMBURGER, F. (ED.). SAFETY EVALUTION AND REGULATION OF CHEMICALS; 1ST INTERNATIONAL CONFERENCE; BOSTON, MASS., USA, FEB. 24-26, 1982. XIV+294P. S. KARGER: BASEL, SWITZERLAND; NEW YORK, N.Y., USA. ILLUS. ISBN 3-8055-3578-3.; 0 (0). 1983. P233-242.
AB- HEEP COPYRIGHT: BIOL ABS. RRM
AB- MOUSE HUMAN INHALED PARTICLES CELLULAR PROLIFERATION PRIMARY DNA DAMAGE IMMUNE FUNCTION ENDOCRINE FUNCTION OCCUPATIONAL EXPOSURE ENVIRONMENTAL POLLUTANTS CIGARETTE SMOKE

5/AU,TI,SO,DE,AB/45 (Item 11 from file: 156)
AU- Wolff RK; Kanapilly GM; Gray RH; McClellan RO
TI- Deposition and retention of inhaled aggregate 67Ga2O3 particles in beagle dogs, Fischer-344 rats, and CD-1 mice.
SO- Am Ind Hyg Assoc J; VOL 45, ISS 6, 1984, P377-81
DE- Aerosols; Body Burden; Dogs; Gallium Radioisotopes --Metabolism --ME; Rats; Rats, Inbred F344
DE- *Gallium --Metabolism --ME; *Lung --Metabolism --ME
AB- Deposition and retention of inhaled 67Ga2O3 aerosols were measured in ten beagle dogs to provide reasonable estimates for human deposition and in Fischer-344 rats and CD-1 mice to estimate lung burdens in small animals frequently used in toxicological evaluations. Aggregated particles of 67Ga2O3, 0.1 micron mass median diameter (MMD), were produced using heat treatment of 67Ga tetramethylheptanedione. Whole-body counting and gamma camera imaging were used to measure deposition. Pulmonary deposition in dogs was measured as 25% of the inhaled particles for the 0.1 micron particles. Tracheobronchial and nasopharyngeal deposition were much lower, 7% and 7% respectively. Pulmonary deposition of 0.1 micron (MMD) particles was calculated as 10% and 11% of inhaled particles in Fischer-344 rats and 15 and 20% in mice for two separate nose-only exposures of each species.

5/AU,TI,SO,DE,AB/46 (Item 12 from file: 156)
AU- CHANG J CF; STEINHAGEN WH; BARROW CS
TI- Effect of single or repeated formaldehyde exposure on minute volume of B6C3F1 mice and F-344 rats.
SO- TOXICOL APPL PHARMACOL; 61 (3). 1981. 451-459.
AB- HEEP COPYRIGHT: BIOL ABS.

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AB- To characterize species differences in sensory irritation to formaldehyde (HCHO) and to determine if tidal volume (VT) compensation paralleled the respiratory rate (f) depression, f, VT and minute volume (VE) of B6C3F1 mice and F-344 rats were measured during 10-min, head-only HCHO exposures. Comparisons also were made between naive (no previous HCHO exposure) and pretreated animals (HCHO, 2, 6 or 15 ppm, 6 h/day for 4 days). HCHO exposure elicited immediate and simultaneous decreases in f and VE in a concentration-dependent manner. No significant compensation in VT was observed in either species except the 15-ppm-pretreated rats. While f and VE remained maximally depressed in mice for the remainder of the exposure period, recovery occurred in rats. In addition to tolerance development, rats were also less sensitive to HCHO. The RD50 (concentration at which f decreased by 50%) of naive rats was 31.7 ppm vs. 4.9 ppm for mice. Similar RD50 values and time-response curves for VE and f were observed among naive and pretreated groups, although the baseline f of pretreated animals was significantly depressed as compared to naive animals. HCHO exposure depressed VE in mice and rats. Because of greater sensitivity and lack of tolerance, mice were able to minimize inhalation of HCHO. This species difference may contribute to differences in respiratory tract toxicity from inhaled HCHO.

5/AU,TI,SO,DE,AB/47 (Item 13 from file: 156)
AU- Kraft LM; Kelly MA; Johnson JE Jr; Benton EV; Henke RP; Cassou R; Haymaker W; Philpott DE; Vogel FS; Zeman W
TI- Effects of high-LET neon (20Ne) particle radiation on the brain, eyes and other head structures of the pocket mouse: a histological study.
SO- Int J Radiat Biol Relat Stud Phys Chem Med; VOL 35, ISS 1, 1979, P33-61

DE- Brain --Pathology --PA Brain --Ultrastructure --UL; Eye --Pathology --PA; Frontal Lobe --Pathology --PA Frontal Lobe --Radiation Effects --RE; Head --Pathology --PA Head --Radiation Effects --RE; Mice; Microscopy, Electron; Nasal Mucosa --Pathology --PA Nasal Mucosa --Radiation Effects --RE; Neuroglia --Pathology --PA Neuroglia --Radiation Effects --RE; Particle Accelerators; Radiation Dosage; Scalp --Pathology --PA Scalp --Radiation Effects --RE; Tooth --Pathology --PA Tooth --Radiation Effects --RE
DE- *Brain --Radiation Effects --RE; *Eye --Radiation Effects --RE; *Neon
AB- A study was made of tissues from 130 pocket mice after a single head-only exposure to high-LET 20Ne particle radiation at 1000, 100 or 10 rad (nominal surface dose) with the view of obtaining base"line data regarding the effectiveness of HZE (cosmic-ray) particles during spaceflight. First seen at 2-3 weeks after exposure, necrotic neurons in the cerebrum reached peak incidence (0 . 04 per cent at 1000 rad, 0 . 003 per cent at 100 rad and less than 0 . 0005 per cent at 10 rad) after 4-5 weeks and decreased to low levels thereafter. Incidence in the cerebellum was lower. Neuroglia, cells of the subependymal matrix and dentate gyrus precursor cells suffered acute damage at 1000 and at 100 rad. At 1000 rad, enlarged hyperchromatic neuroglia, first noted at 3 weeks, increased in number up to 7 months, then declined. Alterations in the retina and olfactory epithelium were seen at 1000 rad, and reaction in the scalp at 100 rad. Damage was incurred by dentinoblasts at 10 rad. Changes similar to those observed in pocket mice were found in the brains of gerbils and C57B1 mice.

5/AU,TI,SO,DE,AB/48 (Item 14 from file: 156)
AU- Eriksen N
TI- Study of the Toxicological Effects of the Inhalation of Gaseous Fluorine at a Concentration of Approximately 25 MG/M exp 3.

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SO- Govt Reports Announcements & Index (GRA&I), Issue 18, 1985
DE- Fluorine, Dogs, Experimental Data, Guinea Pigs, Inhalation, Lun; Toxicity.
AB- TD3: The toxic effects arising from the daily inhalation of gaseous fluorine were studied in 5 animal species. Five dogs, 27 rabbits, 42 guinea pigs, 67 rats, and 49 mice were exposed 6 days weekly for 18 exposure days, a total of 95 hours, to a concentration of fluorine approximating 25 mg/m exp 3 (16 ppM). Nine of the rabbits and 12 of the guinea pigs were subjected to head-exposure only, rather than to the complete exposure to which the majority of the animals were subjected. Toxicity was evaluated from the study of several factors: mortality, growth response, changes in cellular elements of the blood, and tissue damage. The inhalation of fluorine produced 100% mortality in fully exposed rabbits after 32 hours of exposure; in head-exposed rabbits, after 42 hours of exposure; in dogs, after 60 hours of exposure; and in mice, after 93 hours of exposure. At the termination of the experiment of 95 hours of exposure, 50% of the rats, 50% of the fully exposed guinea pigs and 33% of the head-exposed guinea pigs had died. All animals lost weight as exposure progressed, with the exception of the head-exposed guinea pigs, whose average weight remained quite constant. Changes in the number of the red and white blood cells of the dog and rat were observed after a week of exposure, but death ensued before the permanence of the changes were established. In dogs and rabbits significant, consistent pathological changes were seen only in the lungs. These changes were characterized by hemorrhage and edema. Pulmonary damage was also observed in rats, but it was not so consistent, either in severity or nature, as that seen in dogs and rabbits. Testicular degeneration was seen in rats sacrificed at or near the end of the exposure period. In rats, dental fluorosis was manifested grossly after one week of exposure. The amount of fluoride deposited in teeth and bone (incisor and femur) increased with exposure time. 5 refs., 3 tabs. Declassified 4 Dec 1951.

5/AU,TI,SO,DE,AB/49 (Item 15 from file: 156)
AU- Davis JK; Davidson MK; Schoeb TR; Lindsey JR
TI- Decreased Intrapulmonary Killing of Mycoplasma pulmonis after Short-Term Exposure to NO2 is Associated with Damaged Alveolar Macrophages
SO- American Review of Respiratory Disease, Vol. 145, No. 2, pages 406-411, 31 references, 1992
DE- DCN-205332; Nitrogen oxides; Host resistance; Pulmonary clearance; In vivo studies; Laboratory animals; Inhalation studies; Microorganisms; Lung cells

AB- The effect of nitrogen-dioxide (10102440) on pulmonary clearance and intrapulmonary killing of Mycoplasma-pulmonis was studied in mice. C57BL/6N-mice were exposed whole body to 0.5, 1, 2, or 5 parts per million (ppm) nitrogen-dioxide for 4 hours. They were then exposed nose only to aerosols of M-pulmonis that had been tagged with sulfur-35 labeled methionine for 30 minutes. Selected mice were killed immediately or 24 hours after exposure and the lungs were removed. M-pulmonis counts were obtained, from which the degree of pulmonary clearance was determined. Other mice were exposed to 10ppm nitrogen-dioxide and M-pulmonis aerosols as before. Bronchoalveolar lavage (BAL) was performed. Alveolar macrophages in the lavage fluid were separated into the cellular or noncellular fractions or not separated and incubated with M-pulmonis cultures. The extent of cell killing by the macrophages was determined. Mice were exposed to 2, 5, or 10ppm nitrogen-dioxide and M-pulmonis. BAL was performed 1, 3, or 7 days later to determine the effects on lavage fluid cellularity and macrophage viability. Nitrogen-dioxide exposures below 5ppm did not

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